A Critical Analysis of the Relationship Between Parental Smoking and Pulmonary Performance in Children

*R. J. Witorsch, Ph. Witorsch**

Department of Physiology, Medical College of Virginia, Virginia Commonwealth University, Richmond, Virginia
*Departments of Medicine and Physiology, The George Washington University, School of Medicine and Health Sciences, Washington, D.C.

Kritische Analyse des Zusammenhangs zwischen den Rauchgewohnheiten der Eltern und der Lungenfunktion der Kinder

Bis heute sind mindestens 24 Arbeiten über epidemiologische Studien erschienen, in denen die Wirkung derzeitiger oder lebenslanger Rauchgewohnheiten der Eltern auf die Lungenfunktionsparameter ihrer Kinder untersucht wird. In diesen Studien, die sich alle im Ansatz ähneln, wurden die Daten über die elterlichen Rauchgewohnheiten und weitere Angaben, die mit Hilfe standardisierter Fragebogen ermittelt worden waren, mit spirometrischen Messungen bei den Kindern statistisch verglichen unter Berücksichtigung des Raucherstatus der Eltern. Eine Übersicht über diese Arbeiten zeigt eine Anzahl von Widersprüchen bezüglich des Zusammenhangs zwischen dem Raucherstatus der Eltern und den Lungenfunktionsparametern der Kinder (FEV_1 oder $FEV_{0,75}$, FEF_{25-75}, FVC und $V_{max\,50\%}$).

Bei der Interpretation der Ergebnisse sollten eine Anzahl Faktoren in Erwägung gezogen werden, insbesondere im Hinblick auf die festgestellten Widersprüche sowie in Anbetracht der Tatsache, daß die Kinder ausschließlich nach den durch Fragebogen ermittelten Daten eingestuft wurden. Dazu gehören das Mißklassifikationsbias, wodurch die Wirkung des elterlichen Rauchens entweder unter- oder überbewertet wird, der sozioökonomische Status, weitere Variablen sowie genetische Faktoren. Ferner müssen die Wirkung des Rauchens während der Schwangerschaft und der Stillzeit und die Tabakrauchexposition des Kindes als mögliche Ursachen einer meßbaren Verminderung der Lungenfunktion bei Kindern in Erwägung gezogen werden.

Summary

To date, at least 24 epidemiologic research papers, of essentially similar design, have been published on the effects of current or lifetime parental smoking on pulmonary function parameters in children. In these studies, parental smoking and other data obtained from standardized questionnaires and spirometric measurements in children were compared statistically according to the smoking status of the parents. A survey of these reports reveals a number of inconsistencies in the association between parental smoking status and pulmonary function parameters (FEV_1 or $FEV_{0.75}$, FEF_{25-75}, FVC, and $V_{max50\%}$) in the child.

A number of factors should be considered when interpreting the results of these studies, particularly in light of the observed inconsistencies and the fact that children were classified solely on the basis of questionnaire data. Among these are sources of misclassification bias, which could either underestimate or overestimate parental smoking effect, socioeconomic status, other variables, and genetic factors. Also, effects of maternal smoking *in utero* or on lactation, as well as exposure of the child to environmental tobacco smoke (ETS), need to be considered as possible causes of any apparent decrement in pulmonary function in children.

Key words:

Parental smoking – Maternal smoking – Passive smoking – Involuntary smoking – Environmental tobacco smoke – ETS – Pulmonary function – Children

Introduction

The Committee on Passive Smoking of the National Research Council, National Academy of Sciences (1986) and the Surgeon General of the United States (1986) recently reviewed evidence suggesting an association between parental smoking and increased incidence of respiratory symptoms and disease in young children. Currently, it is unclear whether this effect is due to environmental tobacco smoke (ETS) exposure or reflects other factors, such as *in utero* effects of maternal smoking or transmission of infection from parents and/or siblings to the child. Effects of parental smoking on volume-flow indices of pulmonary function have also been examined. Such studies in children are of particular interest because developing lungs may be more sensitive to the effects of environmental pollutants, while exposure to ETS may be particularly intense in children, who may spend 60–80% of their time indoors (*Binder* et al. 1976).

Öff. Gesundh.-Wes. 51 (1989) 78–83
© Georg Thieme Verlag Stuttgart · New York

2023383309

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000

Table 1 Studies of the Effects on Parental Smoking on Pulmonary Function in Normal Children

Published study	Source of subjects	No. subjects (ages)
1. *Tager* et al. 1976	East Boston, Mass.	140 (5–31y)
2. *Tager* et al. 1979	East Boston, Mass.	261 (5–19y)
3. *Weiss* et al. 1980	East Boston, Mass.	238 (5–10y)
4. *Tager* et al. 1983[L]	East Boston, Mass.	715 (4–28y)
5. *O'Connor* et al. 1987	East Boston, Mass.	265 (6–21y)
6. *Ware* et al. 1984	Six U. S. cities	7.112 (6–9y)
7. *Berkey* et al. 1986[L]	Six U. S. cities	7.834 (6–10y)
8. *Hasselblad* et al. 1981	Seven U. S. areas	16.689 (6–13y)
9. *Tashkin* et al. 1984	Los Angeles, Cal.	971 (7–17y)
10. *Ekwo* et al. 1983	Iowa City, Ia.	183 (6–12y)
11. *Vedal* et al. 1984	Western Pa.	3.175 (5–14y)
12. *Spinaci* et al. 1985	Turin, Italy	2.385 (11y[a])
13. *Chen* and *Li* 1986	Shanghai, China	571 (8–16y)
14. *Burchfiel* et al. 1986	Tecumseh, Mich.	591 (10–19y)
15. *Yarnell* and *St. Leger* 1979	Cardiff, Wales	214 (7–11y)
16. *Teculesco* et al. 1986	Vandouvre, France	92 (10–16y)
17. *Tsimoyianis* et al. 1987	Nassau County, N. Y.	193 (12–17y)
18. *Lebowitz* et al. 1987[L]	Tucson, Arizona	353 (5.5–25y)
19. *Leeder* et al. 1976	London, England	454 (5y)
20. *Schilling* et al. 1977	Three U. S. cities	816 (7–18y)
21. *Speizer* et al. 1980	Six U. S. cities	5.842 (6–10y)
22. *Dodge* 1982[L]	Three Arizona towns	472 (8–10y)
23. *Lebowitz* et al. 1984	Tucson, Arizona	271 (13.5y[a])
24. *Lebowitz* 1984	Tucson, Arizona	24 (4–24y)

[L] Indicates longitudinal study, otherwise cross-sectional
[a] Average age

Basic Design and Results of Pulmonary Function Studies in Children

To date, at least 24 epidemiologic research papers, of essentially similar design, have been published on the effects of current or life-time parental (or household) smoking on pulmonary function in children (Table 1). In these studies, parental smoking and other data were obtained from standardized questionnaires, usually derived from or similar to the British Medical Research Council questionnaire. Spirometric measurements obtained in the children, usually normalized, were compared statistically according to the smoking status of the parents. Most of these studies have been cross-sectional in nature, although four have been longitudinal, with the particular objective of assessing ETS effects on lung growth and development (studies 4, 7, 18, 22).

Table 2 presents the results of the findings of these studies as they relate to the effects of parental smoking on two commonly used pulmonary function parameters, forced expiratory volume in the first (or 0.75) second (FEV_1 or $FEV_{0.75}$) and forced expiratory flow between the first and last 25% of the forced vital capacity maneuver (FEF_{25-75}). Of 21 studies which measure FEV_1 or $FEV_{0.75}$, 11 report a small ($\leq 7\%$) decrement in this parameter (studies 1, 4–8, 12–16) associated with parental (usually maternal) smoking, while 10 do not (studies 2, 3, 9–11, 18, 20–23). Of the 11 studies measuring FEF_{25-75}, 8 report a decrement in this measurement in children of smoking parents (usually mothers) (studies 2, 3, 5, 9, 11, 13, 15, 17) while 3 do not (studies 4, 10, 12).

In 10 of these studies, the effects of parental smoking on both FEV_1 (or $FEV_{0.75}$) and FEF_{25-75} in children were measured simultaneously (Table 2). A concurrent

Table 2 Effects of Parental Smoking on FEV_1 ($FEV_{0.75}$) and FEF_{25-75} in Normal Children

Study Number[#]	Effects on FEV_1 or $FEV_{0.75}$	Effects on FEF_{25-75}	Comments
1.	decr.*	not reported	males only
2.	none	decr.	change not statist. signif.
3.	none	decr.*	
4.	decr. 7%*	none	decrement in lung growth over 5 years
5.	decr. 5–7%*	decr. 14–15%*	
6.	decr. 0.6–0.9%*	not reported	
7.	decr. 0.85%*	not reported	predicted decrement in lung growth over 5 years
8.	decr. 0.5–2.0%*	not reported	measured $FEV_{0.75}$
9.	none	decr. 2.5%*	effect in females (12–15y) only
10.	none	none	increased responses to isoproterenol
11.	none	decr. 4%*	measured $FEV_{0.75}$; females only
12.	decr.	none	data not presented
13.	decr. 3%	decr. 6%	assoc. with paternal smoking
14.	decr. 4–5%	not reported	males only
15.	decr. 3%*	decr. 12%*	measured $FEV_{0.75}$; females only; no statistical testing performed
16.	decr. 5%	not reported	males only; decr. attributed to taller children in smoking group
17.	not done	decr.+	not statistically significant; based on increased incidence of children with FEF_{25-75} less than 70% predicted value
18.	none	not done	suggested effect on $V_{max\,50\%}/FVC$*
19.	not done	not done	measured peak flows
20.	none	not reported	
21.	none	not reported	
22.	none	not reported	measured yearly lung growth rates
23.	none	not reported	
24.	not done	not done	measured peak flows+

[#] Numbers correspond to those in Table 1.
* Associated mainly or exclusively with maternal smoking
+ Associated with household tobacco use

decrease in both parameters in children of smoking parents was reported in 3 studies (5, 13, 15). Two studies reported a decrease in FEV_1 without a decrease in FEF_{25-75} (4 and 12), 4 studies reported the converse situation (2, 3, 9, 11), and 1 study (10) reported no effect on either parameter.

2023383310

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000

Table 3 "Dose"-Related Effects of Parental Smoking on Pulmonary Function in Normal Children

Study Number[d]	Parameter	Varies with the no. of Cigarettes	Varies with the no. of Smokers
1.	FEV_1	yes	not avail.
4.	FEV_1	not done	no
6.	FEV_1	yes	no/yes[c]
7.	FEV_1	yes	yes[d]
8.	$FEV_{0.75}$	yes	no
13.	FEV_1	yes	yes
14.	FEV_1	yes[a, b]	yes[a, b]
15.	$FEV_{0.75}$	no	not avail.
2.	FEF_{25-75}	not done	yes[a]
3.	FEF_{25-75}	not done	yes
9.	FEF_{25-75}	no	not avail.
11.	FEF_{25-75}	no	no
13.	FEF_{25-75}	yes	yes
15.	FEF_{25-75}	yes	not avail.

[d] Numbers correspond to those in Table 1.
[a] Concluded on the basis of trends
[b] Males only
[c] In 1 of 2 examinations effect was proportional to number of smokers.
[d] Proportional to number of smokers for FEV level, not for pulmonary growth rate

Table 4 Effects of Parental Smoking on FVC, PEFR, and $V_{max\ 50\%}$ in Normal Children

Study Number[d]	FVC	PEFR	$V_{max\ 50\%}$
5.	none		
6.	incr.		
7.	none		
9.	none		decr.[c]
10.	none	none	none
11.	incr.		
12.	none		none
14.	decr.[a]		decr.[b]
15.	none		
16.	none		decr.[a]
17.	none		
18.	incr.		none
19.		none	
20.	none	none	decr.[b]
21.	incr.		
23.	none		none
24.		none	

[d] Numbers correspond to those in Table 1.
[a] Males only
[b] Females only
[c] Males, 7–11y; females, 12–17y

A number of the papers report the results of studies done at different times on the same population of children (studies 1–5 and studies 6, 7, 21), and reveal inconsistent effects on both parameters, examined either separately or concurrently. Three studies lack statistical verification of the significance of the effects observed (studies 2, 15, 17). In some of the studies, effects are noted in one gender of child and not the other (studies 1, 9, 11, 14–16). In two reports, there were no apparent effects on FEV_1, or FEF_{25-75}, but effects on other pulmonary function parameters were noted (studies 10, 18).

Table 3 presents the reported dose-response relationships between these pulmonary function parameters in children and quantitative estimates of tobacco smoke exposure. FEV_1 (or $FEV_{0.75}$) varied inversely with the number of cigarettes smoked by the parent in six of seven studies (1, 6, 7, 8, 13, 14, 15) and with the number of household smokers in four of six studies (4, 6, 7, 8, 13, 14). FEF_{25-75} varied inversely with number of cigarettes smoked in two of four studies (9, 11, 13, 15) and with the number of household smokers in three of four studies (2, 3, 11, 13). An inverse dose-related effect associated with the number of cigarettes smoked was usually, but not always, accompanied by an effect associated with the number of smokers, where both types of observation were made.

Other pulmonary function parameters in children have also been examined with respect to effects of parental smoking in some of the studies (Table 4). Of the 15 studies which present data on forced vital capacity (FVC), parental smoking is associated with an increase in this parameter in four (6, 11, 18, 21), a decrease in this parameter in males in one (14), and no change in the remaining studies (5, 7, 9, 10, 12, 15–17, 20, 23). No association between peak expiratory flow rate (PEFR) in children and parental smoking was found in the four reports which have measured this parameter (studies 10, 19, 20, 24). When other parameters have been measured, the findings from one study to the next have either been inconsistent, as in the case of $V_{max\ 50\%}$ (Table 4), or have been insufficiently replicated to assess the degree of consistency.

A small number of studies have examined the effect of parental or household smoking on pulmonary function in asthmatic children. Three studies report no apparent effect of parental or household smoking on baseline pulmonary parameters, such as FEV_1, FEF_{25-75}, or PEFR, in asthmatic children (*Tashkin* et al. 1984 [study 9]; *Evans* et al. 1987; *O'Connor* et al. 1987 [study 5]). On the other hand, *Murray* and *Morrison* (1986) report that maternal smoking is associated with a 13% decrease in FEV_1 and a 23% decrease in FEF_{25-75} in asthmatic children. In addition, these investigators observed a fourfold increase in the bronchoconstrictor response to histamine in asthmatic children with smoking mothers. Similarly, *O'Connor* et al. (1987) (study 5) suggest a hyperresponsiveness (ie., increased bronchoconstriction) in asthmatic children of smoking parents to cold air bronchial challenge although, as stated above, no decrement in baseline function was observed in this group. *Ekwo* et al. (study 10) reported increased bronchodilatation in response to isoproterenol in nonasthmatic children of smoking mothers. The significance of these various observations regarding bronchial responsiveness remains to be determined.

Factors Influencing the Outcome of Studies Relating Parental Smoking to Pulmonary Function in Children

The majority of the 24 studies reviewed above (1–18) conclude that parental (usually maternal) smoking produces decrements in pulmonary function in children. However, as pointed out in the preceding section, inconsistencies exist in the specific parameters examined, both between different sample populations and within the same sample population. In the absence of biologic markers (*Jarvis* et al.

2023383311

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000

1985) or some other method of verification, the validity of the questionnaire exposure data in these studies remains to be determined and is, at least, open to some uncertainty. In addition, several factors can influence the results of such studies either "positively" or "negatively", and hence, may contribute to some of the inconsistencies observed in the data. Among these factors are misclassification bias, socioeconomic status, and other variables.

Misclassification Bias

Misclassification errors in the assignment of subjects to groups can create bias that either diminishes or exaggerates effects. One source of misclassification bias is erroneous reporting of parental smoking status. It has recently been estimated that as many as 5 per cent of the respondents in epidemiologic studies who claim to be never smokers are either current or ex-smokers (Committee on Passive Smoking, 1986). Under these circumstances, a child of a misclassified smoker or ex-smoker will be incorrectly classified into the nonsmoking or never smoking parents group, thereby leading to an underestimation of parental smoking-related effects. Similar misclassification bias can result from a change of parental smoking status. It has been reported that smoking status may change at a rate of 1.25 to 2.5% per year (*Berkey* et al. 1986 [study 7]). In studies where subjects are classified on the basis of *current* parental smoking (studies 4–12, 14, 15, 18, 20, 22) rather than lifetime exposure (studies 1, 2, 13, 19, 23), this would also result in the unexposed group containing misclassified exposed children, which would also tend to underestimate parental smoking-related effects. Over an extended period of time, the amount of misclassification error due to change of parental smoking status could be substantial (e. g., as high as 15% in a study of 6 years in duration).

Another source of misclassification bias is lack of consideration or underestimation of active smoking in children. *Kerigan* et al. (1986) recently estimated that 24% of the children between the ages of 8–13 years in a cohort in Hamilton, Ontario reported a history of smoking. A progressive increase in smoking occurred with age, such that at 13 years of age 50% of the children had smoked, with a significant proportion having smoked in the preceding 4 weeks. Other studies estimate tobacco use by children ranging from 2% or less (studies 6, 7, 11–14, 23) to up to 26% (studies 1–3, 5, 9, 17). It has been noted that childhood smoking impairs pulmonary performance (*Tager* et al. 1979 [study 2]; *Lebowitz* et al. 1987 [study 18]). If, as has been implied, there is a greater likelihood that smoking parents will have smoking children (Committee on Passive Smoking, 1986; U.S. Surgeon General, 1986; *Lebowitz* et al. 1987 [study 18]; *O'Connor* et al. 1987 [study 5]), then this form of misclassification would lead to an overestimation of parental smoking effect.

Treatment of active smoking in children varied among the 24 epidemiologic studies reviewed herein. Although some of the studies obtained estimates of childhood active smoking, usually by questioning children in the absence of their parents (studies 1–7, 9, 11–14, 16–17, 20, 23), it was generally assumed, without specific verification, that children below a certain age were nonsmokers. Depending on the particular study, this age ranged from 9 years (studies 6, 7, 11) to up to 15 years (studies 9, 14, 23). In some studies smoking children were excluded from the data base (studies 2, 4, 5, 7, 9, 11, 13, 14, 16–18, 20, 23), while in others they were not (studies 1, 3, 6, 8, 10, 15, 19, 21, 22). In one study, the authors suggested that active smoking in children could account for the apparent effect of maternal smoking on pulmonary function in girls aged 12–17 years (*Tashkin* et al. 1984 [study 9]).

Socioeconomic Status (SES)

It has been shown that the prevalence of parental smoking is inversely related to family income and positively correlated with a number of SES-related factors that potentially can adversely influence childhood pulmonary function, such as poorer outdoor air quality, increased parental coughing, higher gas stove usage, frequent change of address, and lower per capita living space (*Kerigan* et al. 1986). Furthermore, pulmonary test performance in children appears to be influenced by SES, learning, and motivation (*Berkey* et al. 1986 [study 7]; *Dockery* et al. 1983; *Kerigan* et al. 1986; *Ware* et al. 1984 [study 6]). Conceivably, active smoking in children, a source of bias discussed above, may also be inversely related to SES.

Appropriate correction for SES is a matter of debate but appears complex. Among the factors proposed by *Green* (1970) as components of an SES adjustment of a data base in health-related studies are education, income, occupation and ethnicity. In addition, he has suggested that maternal education is more important than paternal education. Some of the studies reviewed here included adjustments for SES (studies 1–4, 6–8, 11–14, 19–21, 23), but others did not (studies 5, 9, 10, 15–17, 22). However, even where adjustments for SES were made, the procedures were not as rigorous as those proposed by *Green*. For example, one method of correction for SES was based on the average educational attainment of both parents (less than high school, high school, more than high school) (studies 6 and 7). Such an adjustment cannot discriminate between a household in which both parents have achieved a modest level of education and one in which one parent was highly educated and the other was not. Furthermore, it may be extremely difficult, solely on the basis of relatively crude estimates of SES, to adjust for SES-related factors that may influence respiratory performance and health, such as family attitudes and practices with regard to nutrition, exercise, and stress management.

Other Factors

Although statistical significance is generally assumed to reflect a real or treatment effect and the statistical methodology is presumed to minimize the possibility of chance effects, statistically significant differences can still be due to uncontrolled factors (biases) and/or chance variation (*Inglefinger* et al. 1983). It has been shown that mean pulmonary performance within a single group of children can vary significantly from one spirometry test to the next (*Kerigan* et al. 1986) without any apparent cause. It is noteworthy that such statistically significant differences are similar in magnitude to most of the small decrements in pulmonary function (generally ≤ 5%) reported in children of smoking parents.

2023383312

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000

Among the other factors which could have influenced the outcome of the above epidemiologic studies are outdoor air quality, home heating, air conditioning and humidity, and occupational exposures (which can be a factor in older children). These may or may not be related to SES. Several studies have noted familial aggregation of pulmonary performance (*Tager* et al. 1976 [study 1]; *Lebowitz* et al. 1984 [study 23]; *Higgins* and *Keller*, 1975; and *Lewitter* et al. 1984) suggestive of a genetic relationship. *Lebowitz* et al. (1984 [study 23]) found that adjustment for a genetic variable, body habitus, eliminated any apparent association between maternal smoking and pulmonary performance in children when comparisons were made on the basis of parent-child pairs.

Another issue relates to the mechanism(s) responsible for an effect of maternal smoking on pulmonary function in children. Attribution of such an effect to ambient ETS is presumptive and should not preclude other important considerations. For example, it has been reported that smoking during pregnancy adversely affects growth and development of offspring (*Abel*, 1980) and that maternal smoking may influence lactation (*Nyboe-Andersen* et al. 1982). Therefore, the possibility that effects of maternal smoking on a child's pulmonary function are mediated by *in utero* or lactational mechanisms should also be considered. The findings of two studies are consistent with this possibility (*Stern* et al. 1987; *Yarnell* and *St. Leger*, 1979 [study 15]).

The dose-response relationship between some pulmonary function parameters in children and amount of parental smoking and/or number of smokers in the household is an extremely important criterion and requires further examination. Although the available dose-response data are consistent with an effect of ETS, they are also consistent with the alternative possibilities of childhood smoking, socioeconomic status, and *in utero* and/or lactational mechanisms. Smoking among children may be more likely to occur where parents are heavier smokers and/or where there are more smokers in the household. Socioeconomic factors affecting pulmonary function may relate quantitatively as well as qualitatively to parental smoking. It is also probable that mothers who smoke more postnatally smoked more during pregnancy and lactation.

Summary and Conclusions

There are a number of factors that must be considered in evaluating the published studies dealing with parental smoking and pulmonary function in children, especially in light of the various inconsistencies noted here in the reported data. These include sources of misclassification bias, socioeconomic status and other variables, and genetic factors. Consideration must also be given to *in utero* and lactational mechanisms, in addition to ambient ETS, as possible explanations of the findings.

Acknowledgements

The authors are grateful to Drs. *Nancy J. Baker*, *Sorell L. Schwartz*, and *S. James Kilpatrick, Jr.* for their helpful critical suggestions during the preparation of this manuscript. The authors also acknowledge their mother for her constant support.

References

Abel, E. L.: Smoking during pregnancy: A review of effects on growth and development of offspring. Hum. Biol. 52 (1980) 593–625

Berkey, C. S., J. H. Ware, D. W. Dockery, B. G. Ferris Jr., and *F. E. Speizer*: Indoor air pollution and pulmonary function in preadolescent children. Am. J. Epidemiol. 123 (1986) 250–260

Binder, R. E., C. A. Mitchell, H. R. Hosein, and *A. Bouhuys*: Importance of the indoor environment in air pollution exposure. Arch. Environ. Health 31 (1976) 277–279

Burchfiel, C. M., M. W. Higgins, J. B. Keller, W. F. Howatt, W. J. Butler, and *I. T. T. Higgins*: Passive smoking in childhood: Respiratory conditions and pulmonary function in Tecumseh, Michigan. Am. Rev. Respir. Dis. 133 (1986) 966–973

Chen, Y., and *W. X. Li*: The effect of passive smoking on children's pulmonary function in Shanghai. Am. J. Public Health 76 (1986) 515–518

Committee on Passive Smoking: Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, D. C.: National Academy Press, 1986

Dockery, D. W., C. S. Berkey, J. H. Ware, F. E. Speizer, and *B. G. Ferris, Jr.*: Distribution of forced vital capacity and forced expiratory volume in one second in children 6 to 11 years of age. Am. Rev. Respir. Dis. 128 (1983) 405–412

Dodge, R.: The effects of indoor pollution on Arizona children. Arch. Environ. Health 37 (1982) 151–155

Evans, D., M. J. Levison, C. H. Feldman, N. M. Clark, Y. Wasilewski, B. Levin, and *R. B. Mellins*: The impact of passive smoking on emergency room visits of urban children with asthma. Am. Rev. Respir. Dis. 135 (1987) 567–572

Ekwo, E., M. M. Weinberger, P. A. Lachenbruch, and *W. H. Huntley*: Relationship of parental smoking and gas cooking to respiratory disease in children. Chest 84 (1983) 662–668

Green, L. W.: Manual for scoring socioeconomic status for research on health behavior. Public Health Rep. 85 (1970) 815–827

Hasselblad, V., C. G. Humble, M. G. Graham, and *H. S. Anderson*: Indoor environmental determinants of lung function in children. Am. Rev. Respir. Dis. 123 (1981) 479–485

Higgins, M., and *J. Keller*: Familial occurrence of chronic respiratory disease and familial resemblance in ventilatory capacity. J. Chronic Dis. 28 (1975) 239–251

Inglefinger, J. A., F. Mosteller, L. A. Thibodeau, and *J. H. Ware*: Biostatistics in Clinical Medicine. New York: MacMillan Publishing Co. (1983) p. 224

Jarvis, M. J., M. A. H. Russell, C. Feyerabend, J. R. Eiser, M. Morgan, P. Gammage, and *E. M. Gray*: Passive exposure to tobacco smoke: Saliva cotinine concentrations in a representative population sample of nonsmoking school children. Br. Med. J. 291 (1985) 927–929

Kerigan, A. T., C. H. Goldsmith, and *L. D. Pengelly*: A three-year cohort study of the role of environmental factors in the respiratory health of children in Hamilton, Ontario. Am. Rev. Respir. Dis. 133 (1986) 987–993

Lebowitz, M. D., R. J. Knudson, and *B. Burrows*: Family aggregation of pulmonary function measurements. Am. Rev. Respir. Dis. 129 (1984) 8–11

Lebowitz, M. D.: The effects of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic and nonasthmatic families. Eur. J. Respir. Dis. 133 (1984) 190–195

Lebowitz, M. D., C. J. Holberg, R. J. Knudson, and *B. Burrows*: Longitudinal study of pulmonary function development in childhood, adolescence, and early adulthood. Am. Rev. Respir. Dis. 136 (1987) 69–75

Leeder, S. R., R. T. Corkhill, M. J. Wysoch, W. W. Holland, and *J. R. T. Colley*: Influence of personal and family factors on ventilatory function in children. Br. J. Prev. Soc. Med. 30 (1976) 219–224

Lewitter, F. I., I. B. Tager, M. McGue, P. V. Tishler, and *F. E. Speizer*: Genetic and environmental determinants of level of pulmonary function. Am. J. Epidemiol. 120 (1984) 518–530

Murray, A. B., and *B. J. Morrison*: The effect of cigarette smoke from mothers on bronchial reactivity and severity of symptoms in asthmatic children. J Allergy Clin. Immunol. 77 (1986) 575–581

2023383313

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000

Nyboe-Andersen, A., C. Lund-Andersen, J. Falck Larsen, N. Juel Christensen, J. J. Legros, F. Louis, H. Angelo, and *J. Molin:* Suppressed prolactin but normal neurophysin levels in cigarette smoking breast-feeding women. Clin. Endocrinol. 17 (1982) 363–368

O'Connor, G. T., S. T. Weiss, I. B. Tager, and *F. E. Speizer:* The effect of passive smoking on pulmonary function and nonspecific bronchial responsiveness in a population-based sample of children and young adults. Am. Rev. Respir. Dis. 135 (1987) 800–804

Schilling, R. S. F., A. D. Letai, S. L. Hui, G. J. Beck, J. B. Schoenberg, and *A. Bouhuys:* Lung function, respiratory disease, and smoking in families. Am. J. Epidemiol. 106 (1977) 274–283

Speizer, F. E., B. Ferris Jr., *Y. M. M. Bishop,* and *J. Spengler:* Respiratory disease rates and pulmonary function in children associated with NO_2 exposure. Am. Rev. Respir. Dis. 121 (1980) 3–10

Spinaci, S., W. Arossa, M. Bugiani, P. Natale, C. Bucca, and *G. de Candussio:* The effects of air pollution on the respiratory health of children: A cross-sectional study. Pediatr. Pulmonol. 1 (1985) 262–266

Stern, B., M. Raizenne, R. Burnett, and *J. Kearney:* Respiratory effects of early childhood exposure to passive smoke. Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Vol. 2 (1987) 22–28

Surgeon General: The Report of the Health Consequences of Involuntary Smoking. Rockville, Maryland. U. S. Department of Health and Human Services, 1986

Tager, I. B., B. Rosner, P. V. Tishler, F. E. Speizer, and *E. H. Kass:* Household aggregation of pulmonary function and chronic bronchitis. Am. Rev. Respir. Dis. 114 (1976) 485–492

Tager, I. B., S. T. Weiss, B. Rosner, and *F. E. Speizer:* Effect of parental cigarette smoking on the pulmonary function of children. Am. J. Epidemiol. 110 (1979) 15–26

Tager, I. B., S. T. Weiss, A. Munoz, B. Rosner, and *F. E. Speizer:* Longitudinal study of the effects of maternal smoking on pulmonary function in children. New Engl. J. Med. 309 (1983) 699–703

Tashkin, D. P., V. A. Clark, M. Simmons, C. Reems, A. H. Coulson, L. B. Bourque, J. W. Sayre, R. Detels, and *S. Rokaw:* The UCLA population studies of chronic obstructive respiratory disease. VII. Relationship beetween parental smoking and children's lung function. Am. Rev. Respir. Dis. 129 (1984) 891–897

Teculesco, D. B., Q. T. Pham, W. Varona-Lopez, J. P. Deschamps, M. Marchand, J. C. Henquel, and *M. Manciaux:* The single breath nitrogen test does not detect functional impairment in children with passive exposure to tobacco smoke. Bull. Eur. Physiopathol. Respir. 22 (1986) 605–607

Tsimoyianis, G. V., M. S. Jacobson, J. G. Feldman, M. T. Antonio-Santiago, B. C. Chutario, M. Nussbaum, and *I. R. Shenker:* Reduction in pulmonary function and increased frequency of cough associated with passive smoking in teenage athletes. Pediatrics 80 (1987) 32–36

Vedal, R. E., M. B. Schenker, J. M. Samet, and *F. E. Speizer:* Risk factors for childhood respiratory disease. Am. Rev. Respir. Dis. 130 (1984) 187–192

Ware, J. H., D. W. Dockery, A. Spiro III, *F. E. Speizer,* and *B. G. Ferris* Jr.: Passive smoking, gas cooking, and respiratory health of children living in six cities. Am. Rev. Respir. Dis. 129 (1984) 366–374

Weiss, S. T., I. B. Tager, F. E. Speizer, and *B. Rosner:* Persistent wheeze: Its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. Am. Rev. Respir. Dis. 122 (1980) 697–707

Yarnell, J. W. G., and *A. S. St. Leger:* Respiratory illness, maternal smoking habit and lung function in children. Br. J. Dis. Chest 73 (1979) 230–236

R. J. Witorsch, Ph. D.
Professor of Physiology

Box 551
Medical College of Virginia
Richmond, Virginia 23298
USA

Buchbesprechungen

Ebel, S., J. Roth (Hrsg): **Lexikon der Pharmazie. Studienausgabe.** Bearbeitet von 17 Autoren. 1987. X, 704 S., 2177 Formeldarstellungen und Abb. (Georg Thieme Verlag Stuttgart · New York.) Kart. DM 88,–. ISBN: 3 13 715201 1

Wenige Monate nach dem Erscheinen des Lexikons der Pharmazie von *Ebel* und *Roth* liegt nun auch eine Studienausgabe des Werkes in kartoniertem Einband vor. Der Kaufpreis konnte dadurch immerhin um 40,– DM gesenkt werden, was die Verbreitung des Nachschlagewerkes insbesondere in den weniger zahlungskräftigen Studentenkreisen mit Sicherheit fördern wird.

Bereits bei der Besprechung der gebundenen Ausgabe wies der Rezensent auf die hohe fachliche Qualität des Lexikons hin, die durch die Mitwirkung von 17 bekannten Vertretern der verschiedenen pharmazeutischen Spezialgebiete verkörpert wird. Die Autoren der Stichworte sind zum großen Teil Hochschullehrer der pharmazeutischen Biologie, Chemie und Technologie sowie der Pharmaziegeschichte, stammen aber auch aus dem Bereich der pharmazeutischen Industrie, der Verwaltung und der Verbände.

Hervorzuheben sind neben den Textbeiträgen die vielen graphischen Darstellungen und die Formelbilder für einen Großteil der im Lexikon aufgeführten Substanzen. Das Lexikon der Pharmazie, ein Spiegelbild eines äußerst breit gefächerten Wissensgebietes, kann neben dem Studenten der Pharmazie auch dem praktisch tätigen Apotheker in Offizin, Krankenhaus und Industrie sowie dem in Wissenschaft und Forschung arbeitenden Pharmazeuten, Pharmakologen und Toxikologen empfohlen werden.

R. Hanpft, Kiel

2023383314

Source: https://www.industrydocuments.ucsf.edu/docs/tqyx0000